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Ruptured pancreatic pseudocyst

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Keywords: pancreas; pseudocyst; somatostatin

Pancreatic pseudocysts are collections of fluid which has escaped from the pancreatic ductal tree disrupted by acute inflammation. Pseudocysts are surrounded by a fibrous tissue wall and lack an epithelial lining. They are prone to serious complications, including rupture, haemorrhage, infection and obstruction of surrounding viscera. Treatment of acute pancreatitis with the peptide hormone, somatostatin has been shown to reduce local complications. We report a unique case of a ruptured pancreatic pseudocyst treated successfully with somatostatin. In addition, this patient developed subcutaneous fat necrosis which is a very rare complication of acute pancreatitis.

Case report

A 37-year-old man presented with a 2 day history of epigastric pain, vomiting and increasing distension. He had

had two previous attacks of acute pancreatitis. The last attack was 18 months previously at which time a pancreatic pseudocyst was diagnosed. During the last 18 months he drank 5 pints of beer per day, prior to this he drank up to 15 pints per day. On examination, he had a distended, diffusely tender abdomen with scanty bowel sounds. His white cell count was 17.5×10^9 ul, serum amylase was 3240 iu/l, and peritoneal amylase was 96 000 iu/l. An abdominal CT scan confirmed gross pancreatic ascites, and ERCP showed a leaking pancreatic duct (Figure 1). A diagnosis of ruptured pancreatic pseudocyst was made.

He was treated conservatively with total parenteral nutrition and octreotide acetate, a somatostatin analogue. Octreotide has been shown to reduce both pancreatic, exocrine and endocrine secretion¹. After 6 weeks of treatment his abdominal distension had disappeared and his blood parameters had returned to normal. The relationship of his serum amylase level to treatment with octreotide is shown in Figure 2. Prior to commencement of somatostatin therapy he developed marked subcutaneous fat necrosis affecting his arms and legs. This resolved after 3 weeks. He was discharged 9 weeks after admission, and was well and asymptomatic 3 months later.

Discussion

Pancreatic pseudocysts are localized collections of pancreatic secretion, lacking an epithelial lining but possessing a clearly defined wall made of fibrous tissue and adjacent viscera². Pseudocysts are more common in alcoholic pancreatitis than gallstone pancreatitis (15% vs 3%)³. A wide spectrum of complications may occur in patients with untreated pseudocysts. The most serious is haemorrhage into the cyst, carrying a mortality rate of 30-60%⁴. Other complications included infection (11% of pseudocysts), obstruction of the



Figure 1. ERCP demonstrating contrast leaking from the pancreatic duct into the peritoneal cavity

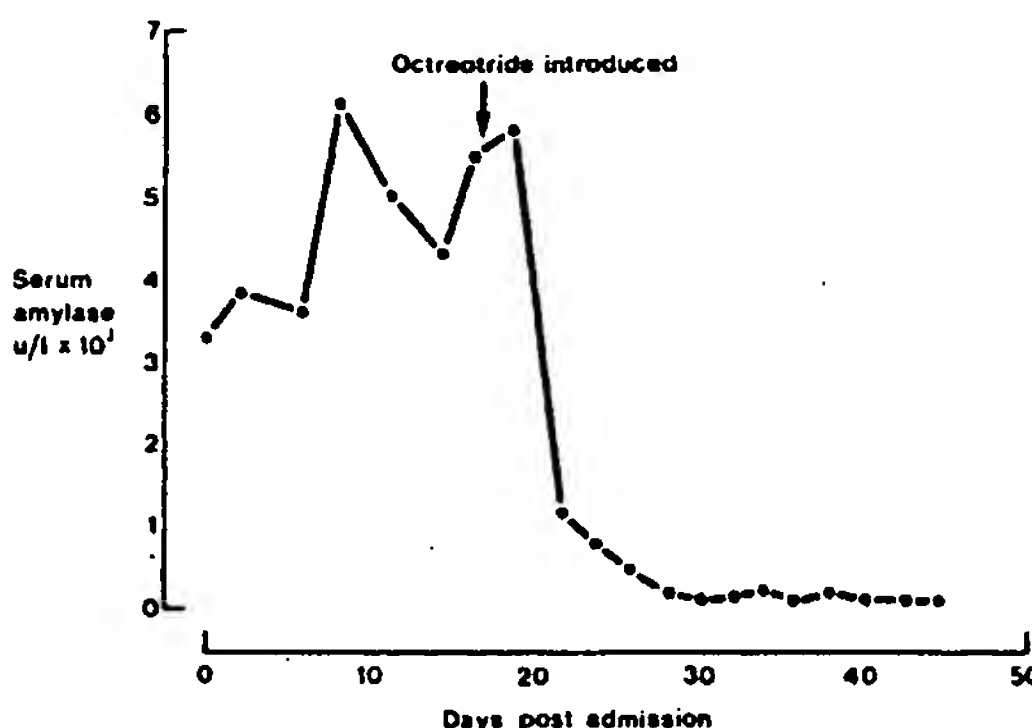


Figure 2. Graph showing the serum amylase changes and the effect of octreotide therapy

Case presented
to Clinical
Section,
8 March 1991

duodenum or rarely common bile duct⁶. A less common complication is spontaneous rupture into the abdominal cavity with a development of pancreatic ascites⁶.

Although it is customary to treat pseudocysts operatively, recent advances have allowed both percutaneous and endoscopic approaches to their management. The peptide hormone somatostatin reduces both exocrine and endocrine pancreatic secretions¹. Recently somatostatin treatment has been shown to reduce the local complications of acute pancreatitis⁶, and to treat external pancreatic fistulae successfully¹.

Therefore it was reasonable to try somatostatin therapy in this case of a ruptured pseudocyst which is, of course, an internal pancreatic fistula. The clinical biochemical and radiological improvements were dramatic. Whilst it is impossible to prove that these improvements were due to somatostatin treatment, it seems highly probable given the temporal relationships of these to the commencement of somatostatin treatment (Figure 2). This is the first report of the use of somatostatin treatment for a ruptured pancreatic pseudocyst.

Another unusual feature of this case was the development of subcutaneous fat necrosis. After pancreatitis, fat necrosis occurring in the mesentery is common. Subcutaneous fat necrosis however is rare, but is well recognized

as a complication of acute pancreatitis⁷. It has unknown aetiology⁷. Nevertheless it was interesting to note the temporal relationship of its improvement with the commencement of somatostatin treatment.

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Neonatal cleft lip repair

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Keywords: cleft lip; ultrasound

A case is reported where a false negative antenatal ultrasound was performed to exclude a cleft abnormality. The reliability of ultrasound is questioned and the role of neonatal cleft lip repair is discussed.

Case report

A newborn male neonate was referred with a diagnosis of a unilateral complete cleft lip and palate. He had no other congenital abnormalities.

The parents' first child had also been born with a bilateral cleft lip and palate and has undergone numerous corrective surgical procedures. Subsequently the mother developed severe postnatal depression, for which she required prolonged psychotherapy. With the onset of the second pregnancy both parents sought antenatal counselling, and ultrasound scans at 16 and 18 weeks (Figure 1) were reported as normal. The parents were reassured and elected to continue with the pregnancy.

At birth, when the cleft defect was apparent, the child was immediately rejected by both parents, with the father in turn rejecting the mother. In view of the fragmentation of the family unit it was decided to repair the cleft lip as a matter of urgency.

The neonate was admitted to Charing Cross Hospital, where he underwent a standard Millard lip repair and was



Figure 1. Sonogram showing view of fetal face in the coronal plane at 18 weeks

transferred back to the maternity hospital the same day. On review 2 weeks later both parents expressed their delight with the operative result, and were reunited as a family.

Discussion

The incidence of combined cleft lip and palate deformities in the UK is stated by Wilson¹ to be 1.47/1000 live births and has a fourfold predominance in males. The predicted recurrence of a cleft defect in children with one affected sibling is 3.2%, and with two affected siblings is 9%².

Only 3% of a clinic cleft population can be linked to identifiable syndromal aetiological factors, such as chromosomal aberrations or teratologic syndromes secondary to drug and alcohol ingestion. The great majority of clefts fall into a 'multifactorial inheritance' category describing a strong familial tendency without Mendelian inheritance patterns.

Case presented
to Section of
Plastic Surgery.
8 May 1991

Ultrasound in the pre-
natal was first popul
in 1981³. However, its
abnormalities has only

Pilu et al.⁶ analysed
craniofacial malformat
of 1% at ultrasound. Fe
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high miscarriage rate
threatening deformity

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it is important to identi
field. High resolution
available, and is cap
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definition of soft tissu
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determined.

Cleft lip repair in ne
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A R Bedford Ru
M D Bain MB ChB
R S Periera MB C
Child Health and
Medical School, Cr

Keywords: mucopolysac
systemic lupus erythe

Two rare disorders
3-year-old Asian boy
(MPSII) and systemi
old male has been de
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Case report

The 3-year-old boy
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developed swollen
intermittent fever.
occasions, when he h
At presentation h
betamethasone.

The parents were
subcontinent. He w
was no family histo
charide disorder.

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